DELAWARE STATE MEDICAL JOURNAL

Official Organ of the Medical Society of Delaware

VOLUME IX NUMBER 9 SEPTEMBER, 1937

Per Year \$2.00 Per Copy 20c

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Entered as second class matter June 28, 1929, at the Post Office at Wilmington, Delaware, under the Act of March 3, 1970. Boolness and Editorial office, 1622 Du Pout Buig., Wilmington, Delaware, Issued monthly. Convirgint, 1937, by the Medical Society of Delaware.

Nutritional Anemia in Infants

THE iron stored in the infant's liver at birth is rapidly depleted during the first months of life (Mackay,' Elvehjem'). During this period the infant's diet contains very little iron—1.44 mg. per day from the average bottle formulae of 20 ounces, or possibly 1.7 mg. per day from 28 ounces of breast milk (Holto). For these reasons, and also because of the low hemoglobin values so frequent among pregnant and nursing mothers (Coons,' Galloway'), the pediatric trend is constantly toward the addition of iron-containing foods at an earlier age, as early as the third or fourth month (Blatt, Glazier, Lynch').

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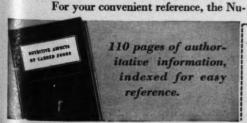
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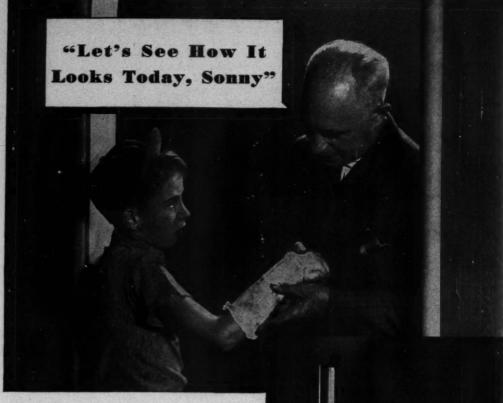
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Laryngoscope, Feb. 1935, Vol. XLV, No. 2, 149-154 ☐

N. Y. State Jour. Med., June 1935, Vol. 35, No. 11 ☐

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1 Cole, Harold N., et al.; J. A. M. A. 108:22, 1937.

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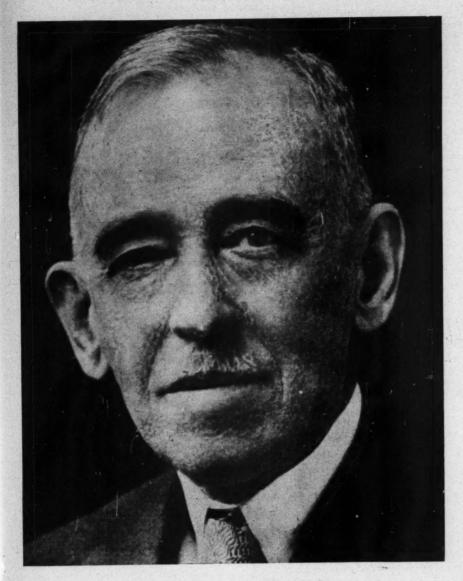
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Owned and Published by the Medical Society of Delaware

Issued Monthly Under the Supervision of the Publication Committee

Volume IX Number 9

SEPTEMBER, 1937

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PREVENTION OF HYPERTENSION*

EDWARD WEISS, M. D.,**

Philadelphia, Pa.

I have taken the liberty of restricting the discussion of this question to essential hypertension, differentiating it from the hypertension so often associated with renal disease. There is little need to emphasize the importance of the subject to this audience. high incidence of cardiovascular renal disease has recently been brought forcibly to our attention by the Metropolitan Life Insurance Company. According to their statistics, every other individual in the United States past the age of 50 years dies of cardiovascular renal disease. From other sources we have evidence that probably half of these deaths are due to essential hypertension, that is, that almost one-quarter of all people past the age of 50 years die of essential hypertension, i. e. of its ill effects on one or another of the vital organs. Thus essential hypertension becomes the gravest problem of middle adult life, not even excepting cancer. As Hedley has stated, "to the individual of 40 the greatest single factor in estimating the probability of living to ripe old age is whether the arterial blood pressure has begun to show signs of elevation."

Now what is the relationship of this disorder to arteriosclerosis and kidney disease? It seems that at last we have pretty well laid the ghost of this hundred-year-old controversy. We know that hypertension precedes arteriosclerosis and not the reverse. We also know that it may be responsible for chronic kidney disease but that essential hypertension is not caused by kidney disease. In this connection I should like to remark that the form

of arteriosclerosis that is due to long standing essential hypertension is a diffuse hyperplastic sclerosis of the vessels, which for clinical purposes may be simply termed hypertensive-vascular disease and that this is quite a different process from atherosclerosis, which is a senile degenerative process affecting principally the large vessels. It may also be stated that the specific end result of lond standing essential hypertension on the kidneys can be referred to as nephrosclerosis, as opposed to chronic glomerulonephritis.

The generally accepted concept of essential hypertension, then, is a vasospastic disorder, widespread in the civilized world, that manifests itself chiefly in middle life, usually runs a course of about 10 to 20 years after its discovery, leads gradually to thickening and constriction of the smaller vessels, especially of the heart, brain and kidneys, so that eventually life is terminated by failure of one or another of these vital organs.

Let us review for a moment what we know concerning the cause of this disorder, because unless we know the etiology of a disease we can hardly hope to prevent it. A schematic outline of our thoughts on this subject might well be represented by a pyramid with three interrelated sides, the base of the pyramid made up of constitutional and hereditary factors and the three sides consisting of vegetative nervous system, the endocrines and the psyche. Stimulation of any one of these systems produces a reaction in the others.

Medicine has always been aware of the importance of constitutional and hereditary factors in hypertension, but it remained for Pearl to confirm our impressions by exact statistical studies in which he proved the high incidence of this disorder in certain families. In considering the vegetative nervous system and its relationship to essential hypertension

^{*}Read before the Sussex County Medical Society, January 14, 1937.

**Professor of Clinical Medicine, Temple University Medical School.

we believe that vasomotor instability is one of the early evidences of hypertension occurring in the early life of the predisposed individual. In regard to the endocrine glands we can speak of the gonads, the adrenals and the pituitary gland. Essential hypertension frequently makes its first appearance at the menopause, and now and again we discover an adrenal body tumor or a basophilic adenoma of the pituitary gland in which hypertension is a prominent symptom. Now this does not mean, of course, that gross derangement of the endocrine glands is to be found in every instance of essential hypertension. does indicate that a rare case occurs in which this is true and it seems to me that it is by a study of the rare cases that we can come to understand better the more common instances of essential hypertension. So far as the psyche of the individual is concerned here, too, we have considerable evidence pointing to its importance. Hypertension frequently makes its appearance with symptoms similar to those of a psychoneurosis. It often appears after long periods of emotional stress and seems to bear a close relationship to anxiety. Moreover, if we make deeper personality studies we often find pronounced evidence of unconscious conflict in the lives of these individuals-conflicts of which they may be totally unaware.

THE RAVAGES OF STRESS

Now, to get a little closer to some of the details of this material, let us assume that if an individual is born with a heavy predisposition toward essential hypertension it will take very little environmental stress to precipitate the condition. On the other hand, if he has little predisposition it might take a very heavy environmental stress to produce the disorder. I have already cited Pearl's studies in regard to constitutional factors and it might now be said, to paraphrase Osler, that we must choose our forebears wisely if we are to avoid hypertension. The young person about to marry would have to choose a mate from a family free of the taint of cardiovascular renal disease. I hesitate to think of the dire consequences that might follow upon the widespread adoption of such advice. In regard to the importance of the environment in the ctiology of essential hypertension a very interesting and important study concerning the incidence of essential hypertension in the American southern negro has just been made. It has been found that essential hypertension is more than twice as frequent in the American southern negro as in the southern white. In this connection it is important to bear in mind that 200 years ago, when the ancestors of the southern negro came to this country, they could not have had essential hypertension since the natives of Africa do not have it today. Therefore, within a period of 200 years the American southern negro by his contact with our civilization has acquired essential hypertension. Consequently it would seem that when we discuss the question of the prevention of hypertension we must direct our attention to the avoidance of stress or at least we must protect against the ravages of stress in the susceptible individual. The susceptible individual can be detected through his heredity, through his evidences of vasomotor instability in early life, such as flushing and palpitation, hyperhidrosis and cephalalgia and by the application of the ice water test of Hines and Brown, a method which determines the reaction of the blood pressure to the introduction of the hand and wrist into ice water.

Now what are these stressful or vasospastic agents the effects of which we must try to minimize? First of all there is the question of obesity, because the build and the weight of the individual have been shown to be of real importance in the etiology of hypertension. I cannot take the time to discuss with you all of the dietary fads that have prevailed in medical thought in regard to the etiology of essential hypertension. We can dismiss the question by saying that in spite of the dread of protein, and red meat in particular, there is no evidence to prove that proteins bear any relation to the cause of essential hypertension, and the same is true of salt, that is, sodium chloride. It is the total diet which is important in regard to obesity.

A second even more important consideration is the question of the toxemia of pregnancy. Recently it has been shown from two well established clinics that women who have toxemia of pregnancy will develop in fifty per cent of instances hypertensive vascular disease or glomerulonephritis within a period of five years. This seems to be true of the milder degrees of toxemia as well as of the more severe forms. The clinical deduction is obvious—if we are to prevent hypertensive vascular disease and kidney disease we must make less effort to nurse these women through their toxemias and must make more effort to interrupt their pregnancies or induce labor.

FOCAL INFECTION

When the chapter on focal infection is finally written we will find that we have been overcredulous in regard to this concept in the etiology of many chronic diseases. I am referring particularly to the question of foci of infection as a clause of essential hypertension. I do not know of any conclusive evidence that there is any real relationship between them, and this is just as true in regard to treatment. The removal of so-called foci of infection will not cure essential hypertension. This does not mean that foci of infection should not be removed from hypertensive or prehypertensive people, but only for the same reasons that they are removed in other people.

Tobacco seems definitely to be a vasospastic agent in some persons and, in my opinion, had better be denied to hypertensives and candidates for hypertension. I do not think the same thing is true of alcohol; I can find no evidence that alcohol is etiologically related to essential hypertension.

The menopause is a period when temporary fluctuations of blood pressure occur and such a fluctuation had better be referred to as a hypertensive state rather than hypertensive disease. This is an important distinction, even though hypertensive disease may often follow the spasmodic blood pressure elevations of the climacteric.

The last consideration for discussion is the question of psychic factors in relationship to essential hypertension. When all is said and done, essential hypertension seems to be the result of the reaction of a predisposed individual to his environment. Therefore, it seems reasonable to study this individual in relationship to his environment rather than to throw up our hands hopelessly because of his predisposition. Now this calls for physical and mental hygiene in its best sense. Mental hygiene does not mean, as is so often the case,

that the physician asks the patient "are you worried about anything?" and is satisfied with a negative reply. This, by the way, is the same physician who will say to the hypertensive patient "stop worrying" or "go home and take it easy." The one approach is just about as ineffective as the other. There is only one method that has any merit and that is to encourage the patient to talk about himself as a person rather than as a medical case. This must be done by skillfully directing the conversation rather than by asking direct questions. This will often succeed in bringing conflicts into the open. One caution for the physician is in order. It is much safer to listen than to talk. Heedlessly giving advice on important emotional factors carries with it many dangers.

"BRINGING THE BLOOD PRESSURE DOWN"

If I may say one word more directly connected with the treatment of essential hypertension, although the philosophy behind it is directly related to prevention. I think one of the greatest faults in the management of hypertension in this country has been the emphasis on "bringing the blood pressure down." We must look upon the blood pressure curve in exactly the same way that we do the fever curve in an acute infectious disease and realize that it is just as wrong to fasten our attention on bringing blood pressure down as it would be on bringing the fever down without understanding and treating the individual who has the blood pressure or the fever. We have paid altogether too much attention to physical measurements in hypertensive disease. While it is undeniably true that the size of the heart as determined by the x-ray, that the height of the blood pressure as determined by the blood pressure apparatus, that the condition of the retinal arterioles as determined by the ophthalmoscope and the state of renal function as determined by the various tests are all absolutely essential to the understanding of the hypertensive individual, they are only the beginning and not the end of the study. We must try to understand the total personality of the individual who has hypertension or who seems destined to develop it. It is the study of emotional factors which may provide us with the key to the successful management of the individual. 1923 Spruce Street

HYPERTENSION AND CEREBRAL MANIFESTATIONS®

ALFRED GORDON, M. D., Philadelphia, Pa.

The problem of hypertension will always be of great interest to the clinician. Many elements enter into the pathogenesis of the condition, among others: arterial resistance, venous flow of blood, frequency of heart beat, the state of the cardiac muscle and of the central and sympathetic nervous systems. The subject of increased blood pressure must be viewed for its better understanding from a physiological, physical, pathological, and clinical standpoint. We are concerned for our present purpose chiefly with the clinical side and particularly with the influence of hypertension on the function of the brain. However, a few remarks concerning the physiology of hypertension are desirable so that the clinical aspects may be better understood. In this relation all types of arterial pressure should be considered, whether temporary or permanent, physiological or pathological. When we consider the occurrence of hypertension in early life before degenerative processes have supervened, we are dealing with hypertonicity of the arterial wall or angiospasm, which may produce much damage to the brain. In this field there is an opportunity to use preventive measures.

Hypertension accompanied by arteriosclerosis presents a different problem because it is associated with a true degenerative process. The cerebral manifestations of hypertension are, therefore, different from the pathogenetic standpoint when they occur at an early or at an advanced age. At an early age the manifestations are due chiefly to angiospasm; at an advanced age to degenerative arterial change.

ANGIOSPASM

That the cerebral circulation passively follows changes in the general blood pressure was demonstrated years ago by L. Hill. However, that local contractions of cerebral blood vessels occur has been convincingly shown. Cushing demonstrated blanching of the blood vessels in the pia with the use of adrenalin. Wiggers observed the same effects with the use of other drugs. In quinine poisoning there

may be temporary contraction of the retinal arteries. Forbes and Wolff have demonstrated contraction of cerebral vessels upon stimulation of the cervical sympathetic nerve and during this reaction the systemic arterial pressure rises. Cobb in a recent experimental contribution showed that the pial vessels may constrict or dilate in response to appropriate stimulation and that obliterating angiospasm occurs when the vessels are strongly stimulated. The possibility of intermittent spasmodic contraction of the cerebral blood vessels is, therefore, an established fact. Physiologists for a long time denied the possibility of constriction of the cerebral vessels basing their opinion upon the fact that the presence of vasomotor nerves in the cerebral blood vessels had not been demonstrated. At one of the recent meetings of the New York Neurological Society, Howe presented moving pictures of his experimental work upon the cerebral circulation. He exposed the cortex of the brain through a small trephine opening by reflecting the dura but leaving the pia-arachnoid intact. Contraction and dilatation of the cerebral arterioles could be plainly seen. Diapedesis, changes in the circulation and even odema, were all distinctly evident. Thus the possibility of angiospasm occurring in the cerebral vessels can no longer be questioned.

Among the many factors which may cause contraction of cerebral blood vessels, hypertension is conspicuous. Clinical observation show that the majority of cerebral attacks occurs in hypertensives. In 1903 Pal called attention to the fact that transitory amaurosis may occur under the influence of increased blood pressure in lead colic, acute uremia and eclampsia. He cites Elschnig, who observed spasm of the retinal arteries in a case of lead poisoning associated with hypertension. Amyl nitrite relieved the condition. In acute alcoholism angiospasm may occur, and with the lowering of blood pressure the phenomenon disappears. In all of these examples the transient character of the spasm indicates an irritation of the cerebral arteries. The hypertension which accompanies it may be called toxigenic. This is the usual type of increased blood pressure which occurs, as mentioned above, in the predegenerative period of the life of the arteries. Herxheimer and Schultz

^{*}Read before the South Branch, Philadelphia County Medical Society, March 25, 1937.

have shown that in this form of hypertension small foci of softening and small hemorrhages are rare, but if they do occur, there is usually hypertension of the entire arterial bed.

Toxigenic hypertonia is not to be confounded with primary hypertonia which is an entirely different disease. The latter is due to the pathological state of the walls of the arteries. We find here usually not only hypertonic arteries, but also hypertrophy of the left ventricle and eventually arteriosclerosis of the renal arteries. Angiospasm of the cerebral blood vessels may occur in both forms of hypertension, but in the toxigenic form there is no sclerosis of the artery but only irritation of the arterial wall. In primary hypertension there are changes in the intima, and thrombosis is the eventual outcome.

With these brief preliminary remarks, let us now consider the clinical picture of both toxigenic and primary hypertension.

GENERAL SYMPTOMS

Irrespective of whether increased blood pressure occurs in the predegenerative or in the degenerative state of the blood vessels, the general nervous symptoms are: fullness and a sense of distention in the head, which is particularly marked during mental work and consequently there is inability to concentrate and continue the mental effort: dizziness and drowsiness; noises in the head, which increase when the patient lies down; attacks of pain in the head; gradual impairment of memory. As the disease progresses the patient develops an emotional state consisting of fear, of impending danger and intense anxiety. Following this preliminary group of symptoms or in the midst of them insults of apoplectic character may occur. They consist of intermittent, temporary or transient attacks of hemiplegia or monoplegia. These attacks of paralysis may or may not be accompanied by aphasia of equally transient character. Instead of complete motor hemiparalysis there may be only hemiparesis or a very slight weakness. Sometimes there may be repeated attacks of paresthesia on one side, and each sensory attack usually leaves a slight feebleness on the same side. This condition may be aptly compared to intermittent claudication. The latter is due to a spasmodic contraction of the arteries when the muscles

of the legs are put into action. The apoplectic insults are caused by a similar angiospasm of the cerebral arteries. The clinical picture of hemiparesis is never complete. Sometimes it is pronounced, sometimes moderate and at other times exceedingly slight. The degree of involvement of the limbs is in some attacks so trivial that only with a very careful test can a slight difference be detected in the power of the affected and unaffected extremities. The number of attacks varies greatly. Some patients have them on rare occasions, once or twice a year; others have them frequently. The purely sensory attacks consist of a sudden tingling in one arm and one leg, but are always followed by motor weakness on the same side.

These attacks are transient. In my experience their duration is never longer than a few days and often they last but a few minutes. In some cases the attacks last several hours. It is interesting to observe that at first the attacks may be short, but almost each subsequent attack lasts longer; and that no matter how short the attack may be, there remains always a certain degree of paralysis varying from slight weakness to complete loss of power. Objectively, in the very mild cases, one may find only a difference in the dynamometric measurements of both hands, a slightly increased patellar tendon reflex on the affected side. The plantar reflex give no response on the affected side, contrasting strikingly with the prompt flexion of the toes on the sound side, or else a distinct extension. These abnormal manifestations in the reflexes were present in my cases from the time of the first attack, even when the latter was of a sensory nature, and persisted even during the intervals between the different attacks. Another observation, worth mentioning, is that very brief attacks occur more frequently than attacks of longer duration.

The characteristic features of the cerebral insults which occur with hypertension are the same in the predegenerative and definitely degenerative states of the arterial walls. However, the larger number of eases occur at the predegenerative stage. Angiospasm may occur under any circumstances which induce irritation of the vessel wall. Hypertension is one of the most powerful factors in producing

such irritation and vascular spasm. In the presclerotic stage, persistently increased blood pressure, per se, is capable of leading to angiospasm of the cerebral blood vessels, as we observe, for example, in toxigenic hypertension. In primary hypertension two factors are concerned, namely, actual anatomic changes and increased pressure. These deductions correspond with the clinical pictures observed during life. Intermittent angiospasm may repeat itself as long as hypertension is maintained, and disappear when the blood pressure reaches a normal level. On the other hand, if the hypertension is maintained, small insults frequently repeated may be the forerunners of more serious vascular accidents. These small insults are due to a temporary diminution of the blood supply, but if frequently repeated they may lead eventually to softening of the brain in the area supplied by the affected vessels. Such a case was described by Lindsay Stevens, who found at autopsy no arterial disease, but necrosis of the surrounding cerebral tissue. It is evident that we are dealing here with an irritable state of the vessel wall maintained by hypertension, which leads to a repeated sudden occlusion of a blood vessel, and secondarily because of local ischemia to suspension of the function of those parts of the brain supplied by the occluded artery. Repeated suspension of the function of the nervous tissue may lead to permanent damage, so that several attacks of hemiparesis may be followed eventually by permanent hemiplegia. In primary hypertension, due to degeneration of the arterial walls, conditions are different. Here there is structural alteration of the arterial walls and when angiospasm occurs, it is more prolonged and eventually hemorrhage or thrombosis, with softening, usually occurs. Permanent paralysis and disability is the result.

In estimating the significance of intermittent apoplectiform attacks in the presence of hypertension, a distinction should be made between the two varieties of increased blood pressure. One, called toxigenic, may occur in the predegenerative stage of the vessel wall; the other, called primary, is associated with degenerative changes in the arteries. In the first type of hypertension, the management will consist of removal cf arterial irritation at

the earliest possible moment. In toxigenic hypertension, for example, prompt removal of the toxic factor and maintenance of a normal blood pressure should be accomplished if possible. In primary hypertension, in which degenerative change of the vessel was is a prominent feature, the influence of hygienic. dietetic, psychic, emotional, toxic and infectious factors must be considered. All of these play a conspicuous role in hastening degenerative changes of the blood vessels, and a knowledge of their importance is valuable in planning preventive measures. When hypertension is established, the occurrence of the cerebral symptoms described above and of apoplectiform attacks, however slight they may be, and more particularly still the intermittent occurrence of such attacks, should be looked upon as evidence of cerebral angiospasm, which may lead eventually to permanent sensorimotor disorders of the gravest nature. Preventive measures carefully planned and rigidly carried out may long postpone these serious consequences.

1900 Locust St.

Medical School Survey

The Council on Medical Education and Hospitals has now completed the survey of medical schools that was begun three years ago. The procedure employed in making the study has been described by Dr. Herman G. Weiskotten,1 and some of the more significant observations were reported to the House of Delegates at Atlantic City.2 Confidential reports in graphic form have been sent to all the schools for the purpose of assisting them in strengthening their respective programs. In response to this stimulus there are already indications of considerable activity, and substantial improvements are being made in standards of admission, in numbers and qualifications of faculty personnel, and in the kind and amount of practical experience comprised in the clinical teaching. While much still remains to be done, the great majority of schools give evidence of healthy growth and development.—Jour. A. M. A., Aug. 28, 1937.

Weiskotten, H. G.: The Medical School Survey, J. A. M. A. 108: 1026 (March 27) 1937.

Report of the Council on Medical Education and Hospitals, J. A. M. A. 108: 2134 (June 19) 1937.

Medical Society of Delaware

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BY-LAWS Article X

Section 1. No address or paper before this Society, except those of the President, invited guests, and orators, shall occupy more than twenty minutes in its delivery; and no member shall speak longer than five minutes, nor more than once on any subject except by unanimous consent.

SECTION 2. All papers read before this Society shah be typewritten, double spaced, with wide margins. Carbon copies will not be accepted. (Authors will please see that their paper contains their name, address, and teaching or hospital position, if any.)

SECTION 3. All papers read before this Society shall become its property. Each paper shall be deposited with the Secretary immediately after reading.

SECTION 4. The deliberations of this Society shall be governed by parliamentary usage as contained in the latest edition of Roberts' Rules of Order, when not in conflict with these By-Laws.

SECTION 5. The Principles of Medical Ethics of the American Medical Association, as contained in the latest edition, shall govern the conduct of members in their relations to each other, to this Society, and to the public.

Essayists will please remember that all papers presented before the Society become the property of the Society and therefore are not to be published or submitted for publication elsewhere than in the DELAWARE STATE MEDICAL JOURNAL.

IF YOU PLEASE

Delegates addressing the House, and members discussing scientific papers will kindly come to the platform and first give their name and city to the steno-typist. Speak slowly and distinctly. Thank you.

MONDAY, OCTOBER 11, 1937 MEETING OF THE HOUSE OF DELEGATES

Delaware Academy of Medicine 8:30 P. M.

- 1. Call to order.
- 2. Roll Call.
- 3. Reading of Minutes of Last Session.
- 4. Appointment of Committee on Nominations.

- 5. Reports of Officers.
 - a. President.
 - b. Secretary.
 - c. Treasurer.
 - d. Councilors.
- 6. Reports of Standing Committees.
 - a. Scientific Work.
 - b. Public Policy and Legislation.
 - e. Publication.
 - d. Medical Education.
 - e. Hospitals.
 - f. Necrology.
- 7. Reports of Special Committees.
 - a. Woman's Auxiliary.
 - b. Cancer.
 - c. Syphilis.
 - d. Tuberculosis.
 - e. Medical Economics.
 - f. Criminologic Institutes.
 - g. Medical Practice Act.
- 8. Report of Delegate to the American Medical Association.
- 9. Report of Representative to the Delaware Academy of Medicine.
- 10. Unfinished Business.
- 11. New Business.
 - a. Resolutions.
 - b. Communications.
 - c. Appropriations.
 - d. Approval of Scientific Program.
 - e. Selection of Meeting Place.
 - f. Miscellaneous.
- 12. Adjournment.

TUESDAY, OCTOBER 12, 1937 GENERAL SESSION

Delaware Academy of Medicine 9:40 A. M.

7:30-9:30 A. M.—Clinics at Delaware, Wilmington General and St. Francis Hospitals.

9:40 A. M.—Invocation.

Rabbi Henry Tavel, Wilmington.

9:50 A. M.-Address of Welcome:

Hon. Richard McMullen, Governor of Delaware.

10:00 A.M.—Urology: Its Relations to General Medicine.

Lang W. Anderson, M. D., Wilmington

10:40 A.M.—Simplified Diabetic Management—A New Regime.

Joseph M. Barsky, M. D., Wilmington.

11:20 A. M.—Practical Points in Relation to Clinical Surgery.

W. Wayne Babcock, M. D., Philadelphia

LUNCHEON

by the

NEW CASTLE COUNTY MEDICAL SOCIETY

Hotel du Pont 12:30 P. M.

GENERAL SESSION
Delaware Academy of Medicine
2:00 P. M.

2:00 P. M.—Pneumonia.

John J. Cassidy, M. D., Wilmington.

2:40 P.M.—The Danger of Pre-operative Delay in Suspected Brain Tumor Cases.

Frederic H. Leavitt, M. D., Philadelphia.

- 3:20 P. M.—Some Aspects of Visceral Syphilis.
- G. Morris Piersol, M. D., Philadelphia.
- 4:00 P.M.—The Causes and Treatment of Uterine Bleeding.
- Brooke M. Anspach, M. D., Philadelphia.
- 4:40 P. M.—Cardiac Diseases in Pregnancy, with Special Reference to Maternal Deaths from Cardiac Diseases.
- Philip F. Williams, M. D., Philadelphia.

EVENING GENERAL SESSION

Delaware Academy of Medicine 8:30 P. M.

8:30 P.M.—Report of the House of Delegates.

9:00 P. M.—President's Address: Some Aspects of Medicine of Today.

Charles P. White, M. D., Wilmington.

9:45 P. M.-Social Hour.

WEDNESDAY, OCTOBER 13, 1937

GENERAL SESSION

Delaware Academy of Medicine 10:00 A. M.

7:30-9:30 A. M.—Clinics at Delaware, Wilmington General and St. Francis Hospitals.

10:00 A. M.-Industrial Law.

Mr. James B. McManus, Wilmington.

10:40 A. M.—The Importance of An Eye Examination in the Fourth Decade of Life.

William Zentmayer, M. D., Philadelphia.

11:20 A.M.—Fourth Tumors of the Mouth and Jaw.

Robert H. Ivy, M. D., Philadelphia.

12:00 Noon-Election of President.

ANNUAL CONFERENCE

of State Secretaries and Editors will be held at the A. M. A. Building, Chicago, November 18-19, 1937. The officers of State and County Medical Societies are invited.

WOMEN'S AUXILIARY to the MEDICAL SOCIETY OF DELAWARE

TUESDAY, OCTOBER 12, 1937 Hotel Du Pont

10:30 A. M.

PRAYER

Greetings from the Advisory Committee, Medical Society of Delaware.

REPORTS OF OFFICERS

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Unfinished Business

New Business

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EDITORIAL

DELAWARE STATE MEDICAL JOURNAL

Owned and published by the Medical Society of Delaware. Issued about the twentieth of each month under the su-pervision of the Publication Committee.

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VOL. IX SEPTEMBER, 1937 No. 9

POLIOMYELITIS

The average number of cases of poliomyelitis for the past five years in Delaware is 5.6, with an average of one death per year. Up to September 14th of this year there have been 7 cases reported, with one death. All of these cases, with the possible exception of 1, had definite paralysis. Draper (1) states that 50 to 80 per cent of all cases do not go on to paralysis: "This means that the apparent size of any given epidemic in terms of paralysis represents only from a fifth to one-half its actual extent." We cannot consider that we have very much of an epidemic in Delaware at the present time.

There is little doubt but that the clinical picture of "abortive" anterior poliomyelitis

and the pre-paralytic febrile stage of the paralytic type are one and the same thing. The diagnosis in the pre-paralytic stage is far from easy. The prodromal symptoms vary somewhat. There is usually a febrile period, the temperature rising seldom higher than 102 to 103 F. and falls quickly in 24 to 48 hours. This may be associated either with a coryza or cough, or with a gastro-intestinal upset usually with a diarrhea and very often vomiting, or simply general malaise and loss of appetite. The child may become restless and apathetic by turns and is often irritable when aroused or handled. The eyes may have a worried, fearful look. At this time there may be slight rigidity of the neck and Kernig's sign may be present. Following the febrile stage there may be a disappearance of all signs and symptoms for a period of 12 to 48 hours. The child then becomes ill again, often with a headache, and paralysis develops. The superficial lymph nodes are usually palpable and enlarged.

Draper says (3): "The spinal fluid in the early hours of the disease is clear, under increased pressure, and contains no cells. Later, in the second or meningeal stage, the cell count rises and the quantity and pressure of the fluid diminishes. The presence of more than 10 cells per cubic centimeter is abnormal, but in some cases the count may be as high as 2,500. At first the cells are multilobular; but at the end of 24 to 36 hours when the globulin content begins to rise, they are for the most part small mononuclears."

To diagnose poliomyelitis in the pre-paralytic stage one has to think of the disease, and a spinal fluid examination must be made to clinch the diagnosis.

The etiology of the disease is thought to be due to a filtrable virus. It can be transmitted from person to person, but the actual mode of spread is not known. There are, no doubt, healthy carriers of this disease. Although some investigators, notably Flexner, believe that the nose is the natural portal of entry of the virus in man, others hold that the gastro-intestinal route may be the pathway for invasion under certain conditions.

The use of nasal sprays, whether they be picric acid and alum as Armstrong used in Alabama in 1936, or 1% zinc sulphate with pontocaine as recommended by Peet, Echols and Richter, are still very much in the experimental stage. Any physician using them should have a colleague spray his cribiform plate with the material before spraying his patients. A rather skilled technique with a special atomizer is required to administer the spray, and the after effects for several days are not too pleasant, as the writer knows from personal experience.

Flexner, (2) in a recent article on Reinfection in Experimental Poliomyelitis, finds that "monkeys which have recovered from an attack of experimental poliomyelitis are subject to reinfection by the nasal route. Reinfection takes place in monkeys that have recovered from mild and severe attacks, and in animals that have been subjected to hyper immunization."

Concerning treatment, most authorities agree that absolute bed rest is essential, repeated lumbar taps are beneficial, and that if paralysis occurs the paralyzed member should be given absolute rest, splinting if necessary, to prevent contractures, with no massage or stimulation of any kind for several weeks. If the paralysis is ascending and respiration is being impaired, then the use of a respirator is imperative. The value of convalescent serum has still to be proven. Vaccines so far are of little or no value.

Delawareans are singularly fortunate in having available the services of a good contagious hospital—the Doris Memorial Unit—where cases and suspected cases from any part of the State are unhesitantly admitted.

It is important that the Wilmington City Board or the State Board of Health be notified of all suspected cases, so that aid in diagnosis may be given if necessary, and that quarantine and isolation can be attempted.

REFERENCES

MISCELLANEOUS Mass Test of Peet Spray

Five thousand Ontario children under 14 are being treated this week, in a vast clinical test, with the Peet zinc sulphate nasal spray—the most hopeful preventive measure yet discovered in the war against infantile paralysis. Fighting an outbreak which has made increasing inroads since June, and which is not expected to reach its peak until the third week in September, Ontario hospitals and clinics are giving this preventive treatment free to children—at the rate of 1,000 a day.

Never tested conclusively since its recent development by Dr. Max Minor Peet of the University of Michigan, the zinc sulphate spray's value as a preventive measure will be known soon as a result of the Ontario experiment.

"Ontario's experiment with 5,000 cases will be of the utmost importance to medical science," Dr. Thomas M. Rivers, director of the Rockefeller Institute Hospital of New York, told a Toronto newspaper over long-distance telephone.

"With animals the Peet spray works beautifully, but with children I can give no real opinion. There have been tests on individual cases but for scientific purposes we need the results of hundreds of cases to have anything conclusive. We shall all be watching Ontario's results with the greatest interest."

Dr. Rivers said a pieric acid spray, which has shown itself much inferior to the Peet spray in tests with animals, had been used last summer on thousands of children in Alabama, and showed some result, despite administration, in most cases, by insufficiently instructed physicians. "The spray is certainly the most hopeful treatment so far," he declared.

Drs. Paul and John Rauch, staff members of the Hagmeier Clinic at Preston Springs, one of the Ontario institutions offering the free nasal spray treatment to children, declare that the spray, while not yet definitely proven a success in prevention of infantile paralysis, is "the only thing yet found of value as a preventive, and should be made a public health measure." Both have made a special study of the spray at Chicago.

Eight hundred children have been treated in five days at Hagmeier alone, Dr. Paul

⁽¹⁾ Draper, George: Infantile Paralysis, New York, D. Appleton-Century Co. 1935.

⁽²⁾ Flexner, S.: Reinfection (Second Attack) in Experimental Poliomyelitis J. Exp. Med. N. Y. 65; 497, April 1937. Abs. J. A. M. A. 108; 1921, 1937.

⁽³⁾ Cecil, Russell L.: Textbook of Medicine, p. 71; Phila.: W. B. Saunders & Co., 1937.

Hauch reports. The method of administering the spray, although requiring special training and instruction, is a simple one. Older children are treated with a nasal speculum and an atomizer. Younger children, somewhat more difficult to handle, are treated with an ordinary atomizer and then held upside down for a moment to allow the spray to penetrate to a sufficient depth into the nostrils.

Official sponsorship of the treatment was urged by Dr. Rauch. "I think it should be taken up as a voluntary measure," he said. "When an epidemic comes along, medical health officers are usually more aware of it than general practitioners, can give first-hand information, and have the facilities. From the standpoint of statistics, it would be valuable, too, as the more we can centralize the treatment the better."

The use of the zinc sulphate spray is not expensive, Dr. Rauch said, amounting to only a third of a cent per child. "It could easily be handled by any civic health authority without financial drain," he pointed out.

"The main essential is some sort of pressure equipment. All hospitals have such electric pressure machines, and they are not expensive to install." The pressure machine is needed, Dr. Rauch said, to enable the spray to reach an effective depth in the nasal passages.

"There is a point between the nose and the brain cavity," he explained, "which is merely a thin plate of bone—the cribriform plate—with numerous perforations. As far as medical knowledge can substantiate, it is the only point at which viruses such as meningitis and paralysis enter the brain. As far as experimental work has shown, it is through the nasal passages that the paralysis virus enters. It lodges on the mucous membrane of the nose and thus enters the blood stream.

"The only scientifically conducted experiments with the nasal spray yet made, those with monkeys, resulted in producing immunity for about 80 to 90% of those treated, while 100% of the other group, untreated, got the disease.

"Naturally, we do not hope for such a high percentage of immunization in children treated. But no matter what the percentage of cases successfully treated—we expect it to be between 25 and 50 per cent—it would still be more than worth while."

Reaction of the nasal spray treatment, Dr. Rauch said, was sometimes swelling of the nasal membranes, headaches, and occasionally stomach sickness. "But this just lasts for a day or at the most two, and then passes off. The reaction is not nearly as bad as that of the picric acid in an alum combination which they used to use, and which was more astringent."

Value of the spray lasts only a comparatively short time, according to Dr. Charles F. Bolduan, public health education director for New York City. "But the treatment," he said, "is comparatively simple, if done by a competent man. It's by all means worth trying so long as it's done right. Dr. Peet's spray and method is by all odds the most hopeful thing we know of so far."

Action of the spray is "simply a mechanical blocking of the olefactory nerve through which the virus gets up into the brain," Dr. Bolduan said. "It just prevents the nerve from letting the virus through."

Ontario's children and parents have responded to the offer of the free spray treatment by hospitals and clinics to such an extent that Toronto's city hospitals have speeded up clinics to take care of 1,000 a day, it was announced.

(Ed. Note: Cf. Delaware State Medical Journal, July, 1937, for abstracts of the Peet treatment.)

International Medical Assembly

The Inter-State Postgraduate Medical Association of North America extends a very cordial invitation to all physicians in good standing to attend the International Assembly of the Association to be held in the City of St. Louis, Missouri, October 18 to 22, inclusive, 1937.

An unusually interesting clinical and didactic program including all branches of medicine and surgery and the specialties has been arranged by the program committee.

In co-operation with the St. Louis Medical Society and the Missouri State Medical Association, and with the active support of the St. Louis Chamber of Commerce and the St. Louis Convention and Publicity Bureau, a most ex-

cellent opportunity for an intensive week of postgraduate medical instruction is offered by a very large group of acknowledged leaders in the profession.

John F. Erdmann, M. D., President.

Medico-Military Training

The ninth annual training course for Medical Department reservists of the Army and Navy will be held at the Mayo Foundation, Rochester, Minnesota, October 3 to 16, 1937. Attendance has become so increasingly popular that it is now necessary to limit enrollment to 200.

The morning hours will be devoted entirely to professional work in special clinics and study groups. Officers in attendance may select the course they wish to follow. The afternoon and evening will be devoted to a medico-military program under the direction of the Surgeon of the Seventh Corps Area (Army) and the Surgeon of the Ninth Naval District (Navy).

This training is on an inactive duty status and is without expense to the government. Enrollment is open to all Army and Navy reservists of the Medical Departments in good standing. Applications should be submitted to the Surgeon of the Seventh Corps Area, Omaha, Nebraska, or to the Surgeon of the Ninth Naval District, Great Lakes, Illinois.

Academy of Physical Medicine

Philadelphia, October 19, 20, 21

The fifteenth annual meeting of the Academy of Physical Medicine will be held at the Hotel Walton, Philadelphia, October 19, 20, 21, 1937. The Academy, which is international in scope, will present a scientific program based on reports of the most recent research and practice of the various specialties. In addition to the lectures, demonstration clinics will be held at the hospitals of the University of Pennsylvania, Jefferson Medical College, and Temple University.

A copy of the program may be had by addressing William D. McFee, M. D., chairman, Committee on Program and Publication, 41 Bay State Road, Boston, Mass.

BOOK REVIEWS

Clinical Urinalysis and Its Interpretation. By Robert A. Kilduffe, M. D., Director of Laboratories, Atlantic City Hospital. Pp. 428, with 40 illustrations. Cloth. Price \$4.00. Philadelphia: F. A. Davis Company, 1937.

This book is designed for the use of the clinician in connection with his office laboratory, and includes all types of analyses which might be performed by him or his technician. The subject matter is up to date. Thus, under concentration functional tests he describes the method of Fishberg rather than that of Mosenthal. The subject of hormone tests is included. Not content with a mere description of technique, he points out the possible sources of technical error and fallacies. The full remarks concerning interpretation of results, which accompany each chapter, are sound, practical, and sensible, and constitute an important feature of the book. Bibliography and index are well arranged. Several of the illustrations, such as those of a urinometer and a Kjeldhal flask, could well have been omitted, because they give one the wrong impression of a valuable book.

The Laboratory Diagnosis of Syphilis. By Harry Eagle, M. D., Past Assistant Surgeon, United States Public Health Service, Washington, D. C. Pp. 440. Cloth. Price \$5.00. St. Louis: C. V. Mosby Company, 1937.

This book considers the theory, technic, and clinical interpretation of the Wassermann and flocculation tests with serum and spinal fluid. Most of the subject matter is of a technical nature, of interest only to the serologist. Fifty-seven pages are devoted to the subject of clinical interpretation, and this phase is so well presented that one wishes it could be reprinted in some form easily available to every practitioner.

Syphilis, the Next Great Plague to Go. By Morris Fishbein, M. D., Editor, Journal of the American Medical Association. Pp. 70, with 11 illustrations and 13 charts. Cloth. Price, \$1.00. Philadelphia: David McKay Company, 1937.

Doctor Fishbein portrays a concise picture of the disease. It covers all types of the malady, together with the laboratory tests, in simple text. It does not equivocate its title. Written especially for the non-medical person, it

should be received and read heartily by the public, for it answers the questions they wish to know.

An Introduction to Dermatology. By Richard L. Sutton, M. D., Professor of Dermatology, University of Kansas, and Richard L. Sutton, Jr., M. D., Instructor in Dermatology, University of Kansas. Third edition. Pp. 666, with 229 illustrations. Cloth. Price, \$5.00. St. Louis: C. V. Mosby Company, 1937.

This book combines the old and new thought in dermatology, and is written primarily for the beginner and student in the specialty. It is well illustrated with photographs. The chapter on syphilis has been brought up to date, especially the old and new treatment in comparative form, which is commendable. The newer advances are capably described and classified, as well as some of the older classifications which have been rearranged. The text is well edited. Thus the book is easy to read. We heartily recommend it to anyone interested in the subject.

Obstetric and Gynecologic Nursing. By Frederick H. Falls, M. D., Professor of Obstetrics, University of Illinois, and Jane R. McLaughlin, R. N., Instructor in Obstetrics, University of Illinois. Pp. 492, with 83 illustrations. Price, \$3.00. St. Louis: C. V. Mosby Company, 1937.

This book, despite the author's statement in the preface that he combines both subjects under one head, really consists of two books in one volume. The part on obstetrics differs much from other books on the same subject. The illustrations are original drawings and in some instances are superior to the usual run found in textbooks of this type. The text is quite comprehensive, and includes the later thoughts in endocrinology as related to obstetrics, and gives a great deal more in actual nursing procedures than most books on obstetrics for nurses. What is particularly outstanding is the chapter on prenatal care, including the duties of the nurse and the nurse's treatment of the prenatal patient in clinics.

The authors have departed from the usual copying of obsolete matter from one book into another and have completely deleted things that are unessential for nurses, with what we think quite an improvement in an obstetrical textbook for nurses. Several chapters are devoted to the duties of the nurse in home de-

liveries, to the care of the newborn, and are quite instructive.

That portion of the book devoted to gynecology, like the obstetrical portion, has original drawings which are an improvement over the usual. The text itself is comprehensive and is quite modern. The book contains an ample glossary.

The authors have succeeded in writing a textbook which is quite superior to the average in its arrangement, illustrations, and content. There should be no difficulty for this book to find its way into most training schools.

The Traffic in Health. By Charles Solomon, M. D.. Assistant Clinical Professor of Medicine, Long Island College of Medicine. Pp. 393. Cloth. Price, \$2.75. New York: Navarre Publishing Company, 1937.

This volume is a semi-encyclopedic treatise on the patent medicine and cosmetic rackets, in which names and figures are given, as well as ample references to the original sources of information. The language used is unequivocal: one never doubts what the author means. We found the book extremely interesting, and have no hesitancy in recommending it to the profession and public alike.

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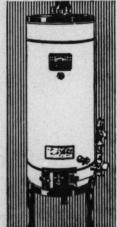
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